

# Circulatory Changes Associated with Systemic Hypotension in Patients with Acute Myocardial Infarction

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Myocardial infarction often leads to a fall in arterial blood pressure. This may occur briefly during the acute stage of the illness, persist for several days, or in some patients, the pressure may fail to return to previous levels. The mechanism by which the blood pressure falls is not always clear, and in order to study the circulatory changes associated with the hypotension, serial hæmodynamic investigations have been made during the acute illness and subsequent recovery in patients with myocardial infarction.

The purpose of this paper is to show that a fall in arterial blood pressure following myocardial infarction can occur in association with widely differing hæmodynamic patterns. The possible physiological and therapeutic implications are discussed.

## SUBJECTS AND METHODS

Investigations are described in 7 patients, all men, admitted to the intensive care unit, and selected to illustrate different patterns of response. Their ages ranged from 47 to 66 years. All had clinical histories and electrocardiographic changes typical of acute myocardial infarction. Three were hypertensive prior to myocardial infarction. One patient (5) suffered from mild diabetes mellitus. All except one recovered and subsequently left hospital.

After clinical assessment, electrocardiography, and chest radiograph, polythene catheters (PE 60 Intramedic, U.S.A.) were introduced percutaneously into the brachial artery and an antecubital vein by the Seldinger technique. The catheters were advanced so that the tip of the arterial catheter lay approximately 10 cm. above the point of insertion and the tip of the venous catheter was in the region of the great veins.

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Intravascular pressure measurements were made with Statham P23Gb transducers with reference to a level 5 cm. below the sternal angle. The patients lay flat with heads comfortably supported on one or two pillows except where otherwise stated.

Cardiac output was measured by an indicator dilution technique using the photoelectric earpiece and Coomassie Blue Dye as indicator (Thomas, Malmcrona, and Shillingford, 1965). Heart rates were measured from an electrocardiographic tracing over half-minute periods recorded at the same time as cardiac output was measured. All the patients were in sinus rhythm at the time of investigation. Hæmodynamic investigations were made in 4 patients (Patients 1, 2, 4, and 5) on each of the first three days, at about one week, and between 3 and 26 weeks after the onset of the acute illness. In Patient 3, measurements were made on the 1st, 6th, and 25th days; in Patient 6, before myocardial infarction and on the 22nd and 50th days afterwards; in Patient 7, on the 1st, 56th, and 141st day.

In the diagrams, the arterial pressures given are direct intravascular measurements, except those indicated by circles which are sphygmomanometric readings. The measurements of cardiac output represent mean values calculated from two or three dye curves, except in Patient 3 where individual measurements are given.

Peripheral resistance was calculated as the mean arterial pressure in mm. Hg divided by cardiac output in l./min. In the case of sphygmomanometric measurements, diastolic pressure plus one-third pulse pressure was used. Oral temperatures were measured with a clinical thermometer, rectal and skin temperatures with an electric thermometer. All hæmodynamic measurements were made with the patient breathing approximately 40 per cent oxygen administered by face mask.

## RESULTS

Hypotension may occur in association with a fall in cardiac output without change in total peripheral resistance, or in association with a fall in total peripheral resistance without change in cardiac output. Variations in arterial pressure may be due to a com-

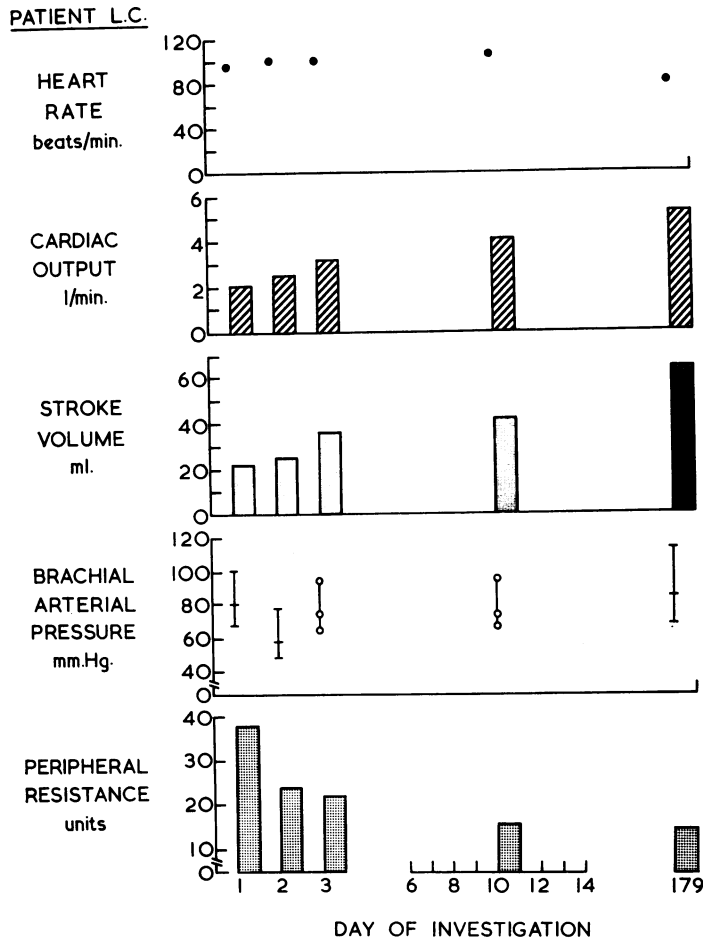


FIG. 1.—Hæmodynamic changes in Patient 1 associated with systemic hypotension and subsequent recovery. Low cardiac output and low stroke volume with high peripheral resistance progressively reverted to normal values in association with the rise in arterial pressure.

bination of cardiac and peripheral factors. These possibilities are illustrated by the study of the following patients.

*Patient 1* (Fig. 1). This 56-year-old man (L.C.) was first studied 14 hours after cardiac infarction and resuscitation following cardiac arrest. At the time of this investigation he was very pale, cyanosed, perspiring, and mentally confused. He was in sinus rhythm, 60/min., with ventricular extrasystoles. Arterial pressure was 100/68 mm. Hg, mean 80. Mean central venous pressure was +5 mm. Hg. The apex beat was not palpable. A third heart sound was present. Râles in the lungs and a pleural rub were audible. No peripheral œdema was present. He was anuric. Rectal temperature was 99°F. (37.2°C.), skin temperature 79°F.

(26°C.). Serum LDH rose to 1500 U. Electrocardiogram showed pathological Q waves and S-T segment elevation in V2-V4. Chest radiograph showed cardiac enlargement and pulmonary œdema.

Cardiac output was 2.1 l./min., stroke volume 22 ml. The peripheral resistance was 38 units. Brachial arterial blood pressure fell from 100/68 mm. Hg, mean 80, on the first day to 78/48 mm. Hg, mean 57, on the second day. Cardiac output rose to 2.5 l./min. and stroke volume to 25 ml. during the same period. Heart rate remained constant. Peripheral resistance fell to 23 units. Oral temperature was normal at the time of the second study. Urine was passed on the second day and clinical improvement progressed uneventfully. This was associated with an increase in cardiac output and stroke volume. Hæmodynamic measurements during

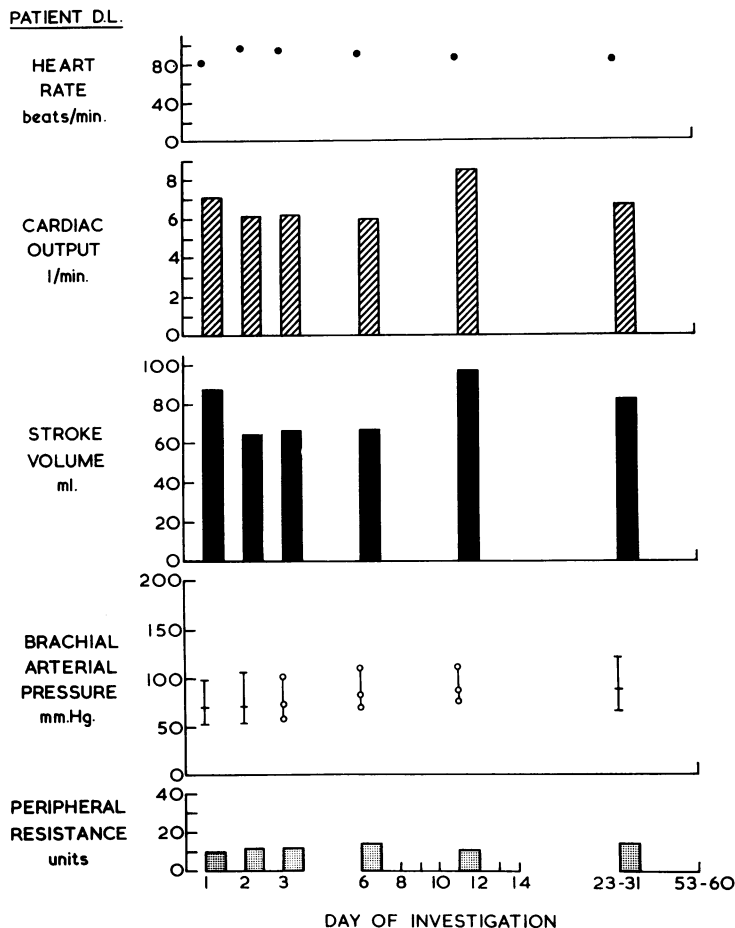


FIG. 2.—Haemodynamic changes in Patient 2 associated with systemic hypotension and subsequent recovery. Initially cardiac output and stroke volume were within a normal range; peripheral resistance was low. Increase in arterial pressure occurred in association with a rise in peripheral resistance, cardiac output and stroke volume remaining of the same order.

convalescence and after the patient returned to work showed further increase in stroke volume and cardiac output. Arterial pressure rose, peripheral resistance remaining within the normal range.

This patient illustrates how systemic hypotension may be associated with low stroke volume and low cardiac output. An additional fall in arterial pressure on the second day was related to a fall in peripheral resistance, the stroke volume and cardiac output rising slightly. Heart rate remained within a narrow range throughout the acute illness. Clinical improvement with rise in arterial pressure occurred in association with a rise in stroke volume and cardiac output.

*Patient 2* (Fig. 2). This 51-year-old man (D.L.) was admitted to hospital following acute chest pain. Soon

after admission he was successfully resuscitated following ventricular fibrillation. The first haemodynamic study was made 18 hours later. At the time of this investigation he felt very weak, looked ill, but was free from pain. The skin was normal in colour and temperature, but was excessively moist. Oral temperature was normal. He was in sinus rhythm, 82/min. Arterial pressure was 98/53 mm. Hg, mean 70. Mean central venous pressure was +2 mm. Hg. The apex beat was abnormally sustained but normal in position. Heart sounds were normal. No added sounds were present. Crepitations were audible in the lungs. No peripheral oedema was present. Serum LDH rose to 1230 U.

The electrocardiogram showed pathological Q waves in leads V4R-V4; S-T segment elevation in V1-V4. Chest radiograph showed slight cardiac enlargement

and pulmonary venous congestion with pulmonary oedema.

Cardiac output was 7.1 l./min., stroke volume 87 ml. Peripheral resistance was 10 units. Arterial blood pressure rose during convalescence in association with a rise in peripheral resistance. Cardiac output remained of the same order, as did stroke volume.

This patient illustrates how hypotension may occur in association with low total peripheral resistance. The rise in pressure that occurred during convalescence was associated with a rise in peripheral resistance.

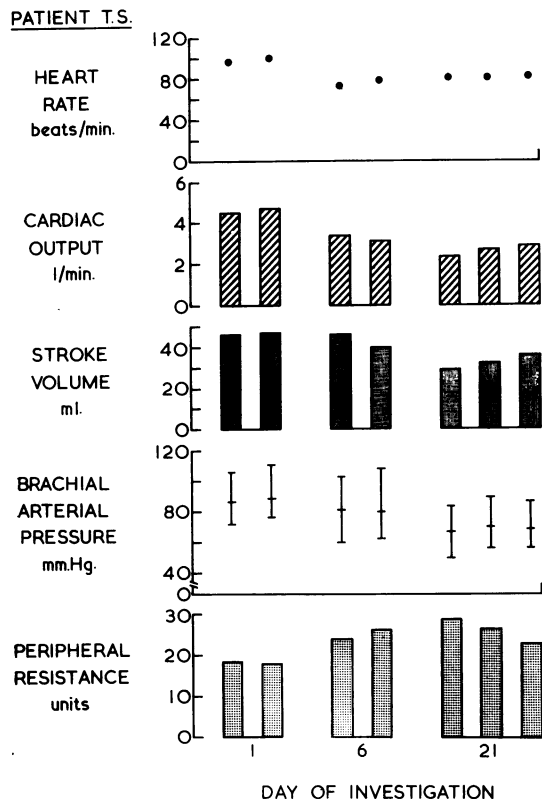


FIG. 3.—Haemodynamic changes in Patient 3 associated with progressive systemic hypotension. Cardiac output and stroke volume fell, the heart rate showing little change. Peripheral resistance increased to a moderate extent.

**Patient 3 (Fig. 3).** This 66-year-old man (T.S.) was admitted following acute chest pain. He looked ill, was pale, cyanosed, and in pain. At the time of the first haemodynamic investigation, heart rate was 100/min. Brachial arterial pressure was 111/78 mm. Hg, mean 89. Mean central venous pressure was 0 cm. of saline. A third heart sound and pericardial friction rub were present. Râles were heard at the lung bases. There was no peripheral oedema. Rectal temperature was 101.3°F. (38.5°C.). The electrocardiogram showed

pathological Q waves and S-T segment elevation in leads V2-V5. Serum LDH rose to 1200 U. Cardiac output was 4.68 l./min., stroke volume 47 ml., peripheral resistance 19 units. During the next few days the patient deteriorated clinically with a fall in arterial pressure. When investigated on the sixth hospital day, heart rate was 74/min., brachial arterial blood pressure was 103/69 mm. Hg, mean 81. Mean central venous pressure was 0 cm. of saline. Cardiac output was 3.38 l./min., stroke volume 46 ml. Peripheral resistance was 24 units. Oral temperature was 98.7°F. (37°C.) at this time; skin temperature was 93°F. (34°C.).

In the following two weeks arterial systolic pressure fell progressively to 80 mm. Hg. A prominent systolic pulsation became palpable over the left præcordium. Skin temperature and appearance remained normal. Chest radiograph showed cardiac enlargement with pulmonary venous distension and some pulmonary oedema. A further haemodynamic investigation was made on the 25th hospital day when the heart rate was 82/min., brachial arterial blood pressure 83/52 mm. Hg, mean 67. Mean central venous pressure was +1 cm. of saline. Cardiac output was 2.4 l./min., stroke volume 29 ml. Peripheral resistance was 28 units. Oral temperature was 97.4°F. (36.4°C.) at this time; skin temperature was 93°F. (33.9°C.). Infusion of metaraminol resulted in the clinical features of acute left ventricular failure and was not continued. Urine output was normal up to the time of death on the 28th day. Necropsy demonstrated a large ventricular aneurysm.

This patient illustrates progressive hypotension occurring in association with falling stroke volume and cardiac output. Peripheral resistance rose only to a moderate extent. Heart rate showed little change in the presence of the fall in arterial pressure and cardiac output.

**Patient 4 (Fig. 4).** This 47-year-old man (J.G.) had had occasional angina for one week before myocardial infarction. Twenty-four hours before admission to hospital on account of a particularly severe chest pain, his blood pressure had been recorded as 160/110 mm. Hg. At the time of the first haemodynamic investigation he looked ill but was free from pain. The skin was pale. Oral temperature was normal. Heart rate was 51/min. Arterial pressure was 131/72 mm. Hg, mean 94. Mean central venous pressure was +7 mm. Hg. An atrial sound was present. The lungs were normal to auscultation. There was no peripheral oedema. The electrocardiogram showed pathological Q waves in leads II, III, and aVF, with S-T segment elevation in V4. Chest radiograph showed slight cardiac enlargement with normal lung fields. Cardiac output was 4.7 l./min., stroke volume 94 ml. Peripheral resistance was 20 units.

During the following three days, arterial blood pressure increased to hypertensive levels (180/120 mm. Hg). This was associated with some rise in cardiac output in relation to an increase in heart rate and also to a progressive rise in peripheral resistance. Oral temperature was normal on the second day and 99.1°F. (37.2°C.) on

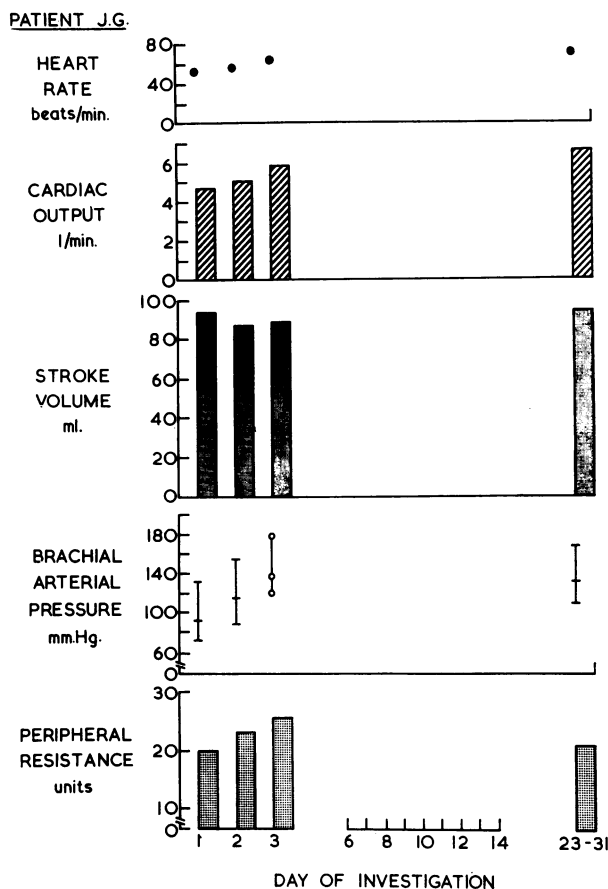


FIG. 4.—Hæmodynamic changes in Patient 4 associated with a rise in arterial pressure to a hypertensive level. The increase in pressure occurred in association with increased heart rate and peripheral resistance. Stroke volume remained of the same order.

the third. Arterial blood pressure remained of the same order during convalescence. At later out-patient visits, it was in the range 180/100/100–120 mm. Hg, before subsequent treatment.

This hypertensive patient illustrates how a fall in arterial pressure may occur in association with a fall in peripheral resistance and a fall in cardiac output due to a low heart rate. Subsequent rise in arterial pressure during the acute illness was associated with a rise in peripheral resistance and heart rate; stroke volume remaining of the same order.

*Patient 5* (Fig. 5). This 60-year-old man (L.Ca) had been noted to be hypertensive before he had myocardial infarction. While attending a diabetic clinic, blood pressure was recorded as 180/100 mm. Hg. He was admitted following a severe chest pain. At the time of the first hæmodynamic investigation he was in pain and restless. Oral temperature was normal. The skin

appearance and temperature were normal. Heart rate was 60/min. Arterial pressure was 196/85 mm. Hg, mean 120. Mean central venous pressure was –2 mm. Hg. An atrial sound was present. The lungs were normal to auscultation. No peripheral oedema was present. The electrocardiogram showed pathological Q waves in leads III and aVF with S–T segment elevation and T wave inversion. Cardiac output was 6.3 l./min., stroke volume 105 ml. Peripheral resistance was 19 units.

During the next three days, when he was free from pain, systolic arterial pressure fell progressively to a level more than 60 mm. Hg below that found on admission. This occurred in association with a marked fall in stroke volume and cardiac output. Peripheral resistance showed little change. Oral temperature was normal during this period. Brachial arterial pressure rose progressively during convalescence in association with an increase in stroke volume and cardiac output.

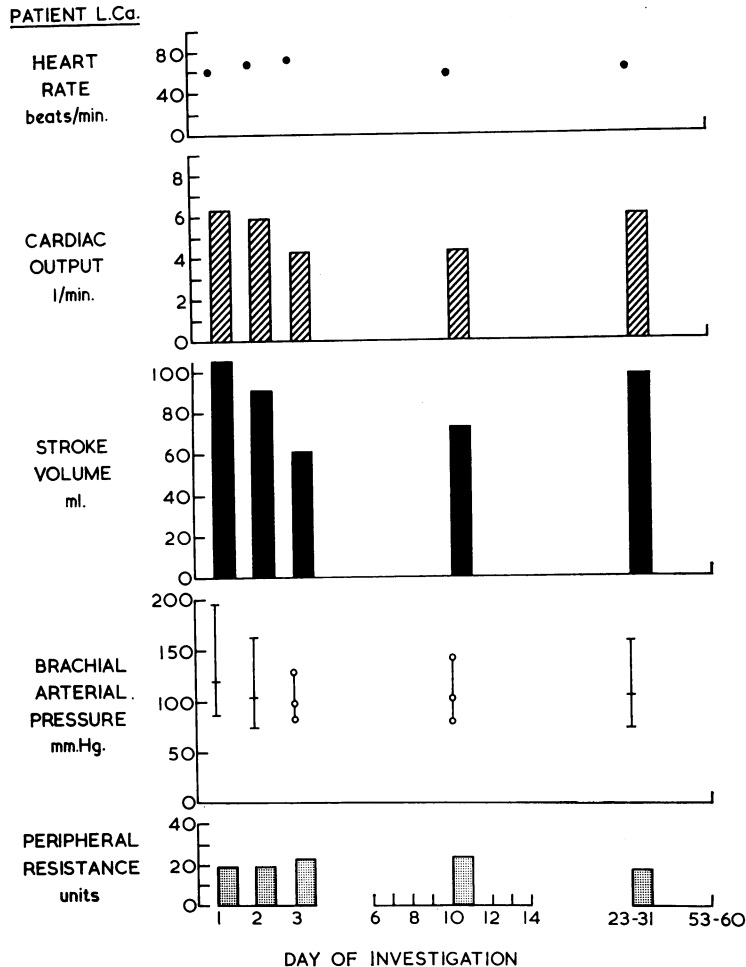


FIG. 5.—Hæmodynamic changes in Patient 5 associated with a fall in arterial pressure from a hypertensive level. Stroke volume and cardiac output fell; heart rate and peripheral resistance rose. Subsequent increase in pressure was associated with an increase of stroke volume and cardiac output.

This previously hypertensive patient illustrates how a fall in arterial pressure may occur in association with a fall in stroke volume and cardiac output, in the presence of a small rise in heart rate and in peripheral resistance. Subsequent increase in arterial pressure did not reach previous levels. The rise in pressure occurred in association with a rise in stroke volume and cardiac output.

*Patient 6* (Fig. 6). This 62-year-old man (J.D.) had been under observation as an out-patient on account of hypertension, mild effort angina, and a history of myocardial infarction in the past. Arterial blood pressure was repeatedly recorded as 180/110–180/120 mm. Hg, using a sphygmomanometer, the last measurement being one month before admission to hospital. He was

in sinus rhythm. The venous pressure was normal. An atrial sound was present.

The electrocardiogram showed S-T changes compatible with ischæmia in leads V4, V5, V6, and aVL. Chest radiograph showed a moderately enlarged heart with evidence of non-specific dust disease of the lungs. Cardiac output measured in a 20° feet down position was 5.2 l./min., stroke volume 72 ml.

Four months after this investigation, during which no change in the clinical condition had occurred, he was admitted following a very severe chest pain and breathlessness. On examination he looked ill, was breathless at rest, and was cyanosed. Oral temperature was 99.8°F. (37.7°C.). Heart rate was 100/min., sinus rhythm. Arterial pressure was 140/90 mm. Hg. Jugular venous

PATIENT J.D.

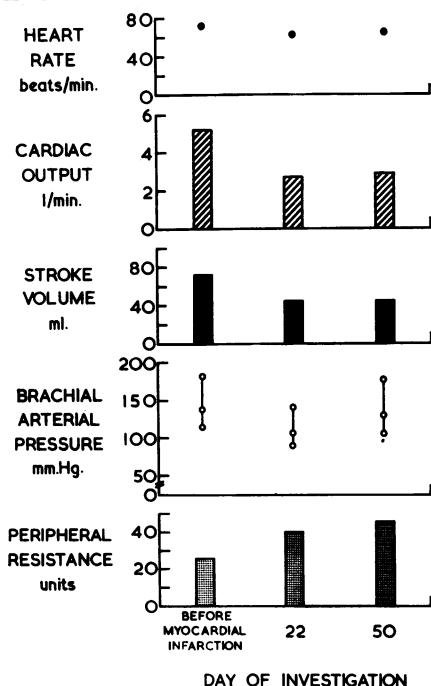


FIG. 6.—Hæmodynamic changes in Patient 6 associated with a fall in arterial pressure from a hypertensive level. A considerable fall in stroke volume and cardiac output occurred in company with a rise in peripheral resistance. Subsequent increase in pressure after several weeks was associated with a rise in peripheral resistance, stroke volume and cardiac output remaining of the same order.

PATIENT T.Sp.

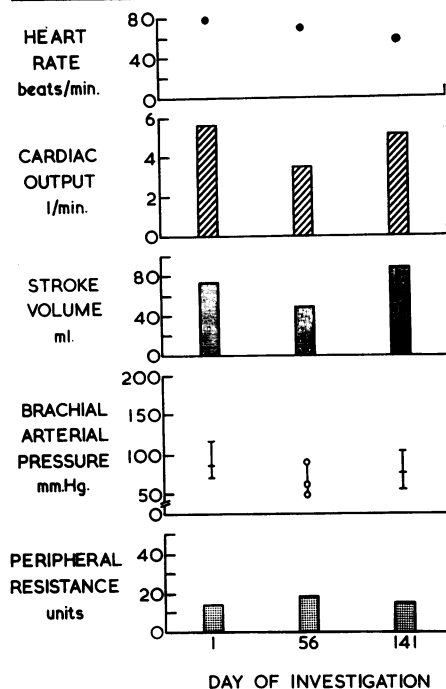


FIG. 7.—Hæmodynamic changes in Patient 7 associated with a fall in arterial pressure from a normotensive level to a hypotensive level. Stroke volume and cardiac output fell. Peripheral resistance rose slightly. Subsequent increase in pressure after several weeks was associated with an increase in stroke volume and cardiac output.

pressure was raised. A third heart sound was present. Crepitations were audible in the lungs.

The electrocardiogram showed S-T segment elevation in leads V2 and V3, with T wave inversion in leads I, aVL, and V4-V6. Chest radiograph showed pulmonary venous distension, interstitial oedema, and further cardiac enlargement as compared with previous films.

He was treated with digitalis and diuretics and improved progressively. Jugular venous pressure returned to normal and the heart became smaller. The third sound was lost but a presystolic sound returned. Arterial pressure did not rise to previous levels and the highest figure recorded was 150/90 mm. Hg. When investigated three weeks after the acute illness, heart rate was 62/min. Arterial pressure was 140/90 mm. Hg. Jugular venous pressure was normal. Cardiac output was 2.7 l./min., stroke volume 43.5 ml.

A further study was made seven weeks after the myocardial infarction when the blood pressure was rising to the range 160/90-170/100 mm. Hg. At this time, heart rate was 64/min., cardiac output 2.8 l./min., stroke

volume 44 ml. The follow-up investigations were made in a 20° feet down position for comparison with the pre-infarction study.

Although the patient made a fair general recovery, exercise tolerance was poor, being limited by angina, fatigue, and breathlessness. He was unable to return to his work as a carpenter and died at home five months after the acute illness. There was no necropsy.

This patient illustrates hæmodynamic changes which may occur when a previously hypertensive patient shows a persistent reduction in arterial pressure following a myocardial infarction. Cardiac output and stroke volume were reduced to 50 per cent of the initial level. When arterial pressure ultimately began to increase, this occurred in association with a rise in peripheral resistance, the stroke volume and cardiac output remaining low.

*Patient 7* (Fig. 7). This 58-year-old man (T.Sp.) was admitted following a severe chest pain and breathlessness while walking. When the first hæmodynamic

investigation was made, pain was only moderate and his general condition was good. Oral temperature was 99.2°F. (37.3°C.). Skin appearance and temperature were normal. Heart rate was 78/min. Brachial arterial pressure was 117/72 mm. Hg, mean 87. Mean central venous pressure was +1 cm. of saline. An atrial sound was present. The lungs were normal to auscultation. No peripheral oedema was present.

The electrocardiogram showed pathological Q waves in leads V3 and V4, I, aVL, with S-T segment elevation in V2-V5, I, aVL. Chest radiograph showed slight cardiac enlargement with normal lung fields. Serum LDH rose to 1050 U. Cardiac output was 5.7 l./min., stroke volume 73 ml. Peripheral resistance was 13 units.

The acute illness was uneventful, but during the first week it was noted that the systolic blood pressure was persistently 90 mm. Hg. After he left hospital, exercise tolerance was fair and there were no symptoms referable to postural hypotension. When a further haemodynamic study was undertaken on the 56th day, heart rate was 70/min., cardiac output 3.46 l./min., stroke volume 49 ml. Arterial blood pressure was 90/50 mm. Hg. Peripheral resistance was 18 units. At this time and at subsequent investigations, the patient was receiving digoxin 0.25 mg. b.d. In the following weeks some general improvement in exercise tolerance was reported by the patient. Haemodynamic study on the 141st day showed a heart rate 58/min., cardiac output 5.05 l./min., stroke volume 87 ml., brachial arterial blood pressure 103/57 mm. Hg, mean 77. Peripheral resistance was 15 units. Measurements of systolic arterial pressure by sphygmomanometry gave figures similar to those obtained by direct intravascular measurement.

This patient illustrates the haemodynamic changes which may occur when persistent hypotension follows myocardial infarction in a previously normotensive patient. At the time when arterial pressure was reduced stroke volume and cardiac output were approximately two-thirds the level measured during the initial stages of the acute illness. Subsequent increase of pressure occurred in association with a rise of stroke volume and cardiac output.

#### DISCUSSION

Hypotension is an outstanding feature of cardiovascular failure in patients with acute myocardial infarction. A low level of blood pressure is often used as an index of the patient's clinical condition and as a guide to the necessity for therapeutic support. Many drugs have been used in treatment and also intravenous and intra-arterial transfusions, but it is recognized that the increase in blood pressure, which is often possible, is not necessarily accompanied by general clinical improvement (Binder *et al.*, 1955). On this account the circulatory changes which accompany systemic hypotension in these patients require study.

It has been shown by haemodynamic studies in man (Grishman and Master, 1941; Pritchard and Hellerstein, 1950; Freis *et al.*, 1952; Gilbert, Goldberg, and Griffin, 1954; Smith, Wikler, and Fox, 1954; Gammill *et al.*, 1955; Lee, 1957; Broch *et al.*, 1959; Murphy *et al.*, 1963) that myocardial failure, as indicated by low stroke volume and cardiac output, is the most common abnormality associated with systemic hypotension in patients with myocardial infarction. The clinical features of skin pallor and tachycardia found in some severely ill patients might suggest that these are compensatory responses secondary to systemic hypotension and small stroke volume. However, the circulatory changes accompanying a fall in arterial pressure in the patients studied by us did not always allow explanation by a simple homeostatic mechanism.

Patient 1 gives an example of the common association of low arterial blood pressure with low stroke volume and cardiac output. The peripheral resistance was very high. The further fall of 20 mm. Hg in arterial pressure between the first and second days was apparently related to a fall in peripheral resistance rather than stroke volume. Cardiac output and stroke volume increased slightly. During convalescence progressive increase of arterial blood pressure was associated with a rise of stroke volume to approximately three times the initial level, and of cardiac output in almost the same proportion. Peripheral resistance returned to the normal range.

This haemodynamic pattern is in marked contrast to that found in Patient 2 in whom the same low initial arterial blood pressure was associated with a stroke volume and cardiac output three times as great as in Patient 1, and within the normal range. The peripheral resistance was low. Rise in blood pressure during convalescence was related to a rise in peripheral resistance. The clinical features shown by this patient when the blood pressure was low were of note, in that the skin was not excessively pale, was of normal temperature, but was sweating excessively.

Patient 3 is an example of progressive cardiac muscle failure which was associated with a fall in cardiac output to 50 per cent of the initial level over a period of three weeks. The extent of muscle damage was indicated by the post-mortem demonstration of a large ventricular aneurysm. In the presence of increasing hypotension the heart rate showed little change. Peripheral resistance rose only to a moderate extent, though systolic arterial blood pressure fell to approximately 80 mm. Hg. This patient is of particular interest in that hypotensive cardiovascular failure, essentially due to defective myocardial function with low cardiac out-



put, was not accompanied by pallor or coolness of the skin.

A fall in arterial blood pressure following myocardial infarction is frequently seen in patients who previously have been hypertensive. That this may be related either to changes in peripheral resistance or to myocardial failure is seen from the measurements made in Patients 4 and 5. Patient 4 was admitted with systolic arterial pressure 50 mm. Hg below the level reached after three days and which ultimately persisted until treatment was given. The increase in arterial pressure was associated with a rise in peripheral resistance and a small rise in cardiac output due to an increase in heart rate. A comparable fall of blood pressure in another previously hypertensive patient (5) was associated with a fall of 30 per cent in stroke volume. Cardiac output fell in almost the same proportion. Peripheral resistance rose slightly. Subsequent increase of blood pressure to previous levels was associated with an increase of stroke volume and cardiac output.

In Patients 1, 2, 4, and 5 hypotension occurred for a varying period during the acute illness but the pressure returned either to normal levels or, in those patients previously hypertensive, to high levels during the period of the acute illness. The return of blood pressure to previous levels appeared to be associated either with changes in peripheral resistance or with changes of stroke volume and cardiac output.

It is known that in some patients blood pressure may fail to return to previous levels. This may be so in hypertensive or normotensive patients. The mechanisms responsible for this are of some interest, and two patients (6 and 7) show examples of the haemodynamic changes which may accompany a persistent fall in blood pressure. In Patient 6 the loss of hypertension for many weeks was related to a 50 per cent reduction in stroke volume and cardiac output. The figures are significant in that a measurement of cardiac output and stroke volume had been made prior to myocardial infarction. When the blood pressure ultimately began to increase, this was related to an increase in peripheral resistance, the stroke volume and cardiac output remaining constant at the low level. Patient 7 was normotensive prior to myocardial infarction and, though serious hypotension did not occur during the acute illness, low levels of systolic blood pressure (90 mm. Hg) were consistently found during convalescence. In this patient also, the stroke volume and cardiac output were found to be reduced to approximately two-thirds of the values found at an early stage in the acute illness when the systolic blood pressure was 115 mm. Hg. Subsequent

increase of arterial pressure was associated with increase of stroke volume and cardiac output.

The factors that govern the type of circulatory response to myocardial infarction are not known, and one may only speculate on the possible physiological mechanisms that may be responsible for some of the haemodynamic patterns associated with systemic hypotension. In some patients it seems clear that the primary and most important factor is failure of the myocardium with resultant low stroke volume and cardiac output. The occurrence of a low pressure state in relation to a low peripheral resistance and high cardiac output may have particular significance with respect to those hypotensive cases with a low cardiac output and a normal or only moderately raised total peripheral resistance. It could be that in these patients the net result includes a combination of vasoconstrictor and vasodilator mechanisms. Measurement of regional flow is necessary to clarify this. Although an increase in the patient's temperature is known to be responsible for a lowered peripheral resistance (Malmcrona and Varnauskas, 1964), this particular factor does not provide a complete explanation for low or falling peripheral resistance in the presence of low arterial pressure, or for those patients in whom total peripheral resistance is only moderately raised when arterial pressure and cardiac output are low.

It might be thought that systemic hypotension would promote an increase in heart rate by means of the carotid sinus and aortic reflex mechanisms. In Patient 3, not only was the heart rate normal in association with hypotension, but showed little change as hypotension increased. In another (4) a lowered arterial pressure occurred in association with a low heart rate which subsequently increased in company with the rise in arterial pressure.

Some experimental work may be relevant to the anomalous circulatory behaviour seen in some of these patients. Dawes (1947) has shown that reflexes initiated from the left ventricle may decrease heart rate and produce a fall in arterial pressure. That such reflexes may induce peripheral vasodilation in animals during occlusion of a coronary artery has also been demonstrated (Costantin, 1963). Whether such reflexes have any place in the physiological changes following myocardial infarction in man remains to be shown.

The results of these haemodynamic studies may have some importance in the practical management of patients who have a fall in arterial pressure following myocardial infarction. In some patients hypotension does not appear to be incompatible with an adequate regional blood flow, and pressor therapy may not be required. The mechanism by which an increase in blood pressure is achieved by

pressor drugs may be different in different patients, not necessarily with equal benefit. It has been observed that metaraminol may increase arterial pressure in patients with acute myocardial infarction by an increase of cardiac output, or of peripheral resistance, or both, the mechanism not bearing any clear relation to the factor apparently responsible for the low blood pressure (Malmcrona, Schröder, and Werko, 1964). In view of these several considerations, it seems necessary to evaluate further the indications for, and effects of, pressor therapy in hypotensive patients with different haemodynamic features.

## SUMMARY

Serial measurements of heart rate, cardiac output, and arterial pressure have been made in patients with acute myocardial infarction admitted to an intensive care unit. Seven patients were selected to illustrate different haemodynamic patterns accompanying a fall in arterial pressure. Low arterial pressure may be associated with low stroke volume and cardiac output or with low peripheral resistance. In some patients both cardiac and peripheral factors may be important. The behaviour of heart rate and peripheral resistance sometimes did not follow a simple homeostatic principle. Persistent low arterial pressure was associated with low stroke volume and cardiac output. Possible physiological and clinical implications of the results are discussed.

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